Serum Leptin And Polycystic Ovary Syndrome: A Brief Review

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Abstract

Polycystic Ovary Syndrome (PCOS) is a prevalent endocrine disorder characterized by reproductive and metabolic complexities. This review investigates the role of serum leptin levels and their relationship with PCOS through an extensive analysis of existing literature, the review explores the intricate role of leptin in PCOS pathophysiology, highlighting its regulatory impact on reproductive hormones and metabolic pathways. Leptin, a multifaceted adipokine, emerges as a significant player in the complex web of PCOS pathophysiology. Based on a comprehensive analysis of current scientific research, the review elucidates the intricate signaling pathways involved in the complex interplay between leptin and polycystic ovary syndrome (PCOS). The synthesis of findings contributes to a deeper understanding of the intricate relationship between serum leptin levels and PCOS, providing valuable insights for the development of targeted diagnostic approaches and therapeutic interventions. This exploration of the predictive significance of serum leptin levels not only enhances our comprehension of PCOS pathophysiology but also holds promise for advancing diagnosing strategies in clinical practice.

Index Terms: PCOS, Leptin, Metabolism, Women health, Reproductive Health
I. INTRODUCTION

Polycystic ovary syndrome (PCOS) is a prevalent endocrine disorder affecting women throughout their reproductive age, from adolescence to post-menopause (Teede et al., 2023). The complex nature of PCOS involves disruptions in both the endocrine and metabolic systems, resulting in various clinical manifestations such as anovulation, infertility, obesity, insulin resistance, and the presence of polycystic ovaries (Singh et al., 2023).

Several risk factors contribute to the susceptibility of females to PCOS. Lifestyle choices, including diet and exercise, play a crucial role, along with exposure to environmental pollutants that may disrupt hormonal balance. Genetic predisposition is also a significant factor, with certain genetic traits increasing the likelihood of developing PCOS. Additionally, disturbances in gut microbiota, known as gut dysbiosis, and neuroendocrine shifts further contribute to the intricate etiology of PCOS (Singh et al., 2023).

The development of metabolic syndrome in PCOS involves several interrelated conditions. Hyperinsulinemia, characterized by elevated insulin levels, is a key feature, leading to insulin resistance. This insulin resistance, in turn, contributes to oxidative stress, a condition marked by an imbalance between reactive oxygen species (ROS) and antioxidant defenses. Hyperandrogenism, involving elevated levels of male sex hormones (androgens), is another prominent aspect of PCOS. This hormonal imbalance affects folliculogenesis, the process of follicle development in the ovaries, leading to impaired ovulation and irregular menstrual cycles (Singh et al., 2023).

This comprehensive understanding of PCOS underscores the intricate interplay between genetic and environmental factors in its pathogenesis. The multifaceted nature of PCOS necessitates a holistic approach to diagnosis and management, considering the diverse factors contributing to its development. Ongoing research continues to unravel the complexities of PCOS, providing insights into potential therapeutic interventions and diagnostic strategies (Teede et al., 2023).

Despite extensive research, the exact underlying causes of PCOS remain elusive. Unraveling the pathogenesis of PCOS requires understanding the intricate relationship between environmental influences and genetic predisposition, paving the way for more effective diagnostic and treatment strategies. Leptin, a multifunctional adipokine, plays a crucial role in energy homeostasis and is implicated in various physiological processes, including reproduction and metabolism (Zheng et al., 2017).

PCOS elevates the risk of diabetes, cardiovascular diseases, and metabolic syndrome in affected individuals (Namavar et al., 2017). Addressing insulin resistance in PCOS necessitates the identification of new markers. Several proteins, such as adipocytokines (adiponectin, visfatin, vaspin, and apelin), copeptin, irisin, PAI-1, and zonulin, have emerged as potential indicators strongly associated with PCOS physiopathology and insulin resistance (Polak et al., 2017). However, proteins like resistin, leptin, RBP4, kisspeptin, and ghrelin proposed as markers face controversies in their roles (Polak et al., 2017).

This review focuses on elucidating the intricate relationship between leptin and PCOS pathophysiology, emphasizing leptin's potential as a diagnostic indicator. By examining the interconnection between leptin, obesity, and the metabolic and hormonal irregularities in PCOS, the aim is to contribute to the existing body of knowledge, exploring the nuanced role of leptin in PCOS development and progression. The ultimate goal is to consider leptin's potential utility as a diagnostic marker in the clinical context, offering insights into the diagnostic precision required for effective PCOS management.

THE RELATIONSHIP BETWEEN LEPTIN AND PCOS

Obesity is prevalent in individuals diagnosed with polycystic ovary syndrome (PCOS), and the hormone leptin is believed to play a crucial role in its pathophysiology, particularly within the context of obesity and oxidative stress (Shetty et al., 2022).
Leptin, belonging to the "tumor necrosis factor" family, is an amino peptide composed of 167 amino acids and is produced by adipose cells in fat tissues. The levels of leptin exhibit a positive correlation with the quantity of fat cells, primarily functioning to regulate appetite and facilitate heat generation for weight control in humans. Elevated leptin levels stimulate the appestat, a brain region regulating food intake, potentially leading to reduced heat generation (Friedman 2014) (Shetty et al., 2022). Leptin's regulatory role in appetite and energy expenditure highlights its significance in the complex interplay of factors contributing to obesity in individuals with PCOS.

Beyond weight regulation, leptin (LEP) has modulatory effects on the maturation of female egg cells and activates ovarian enzymes involved in steroid production, including Follicle-Stimulating Hormone (FSH) and Luteinizing Hormone (LH). This dual role underscores leptin's importance in the intricate pathophysiology of PCOS (Zhao et al., 2023). Ongoing research aims to elucidate precise mechanisms through which leptin operates in the context of PCOS, providing insights for potential therapeutic interventions and diagnostic strategies.

The relationship between leptin and PCOS is subject to ongoing research and controversy. Recent studies suggest that increased intra-follicular levels of leptin in obesity may impact ovarian functions in PCOS, potentially inducing relative resistance to gonadotropins. This emphasizes the intricate connection between leptin, obesity, and hormonal dysregulation characteristic of PCOS (Al-yasiry et al., 2022). Leptin emerges as a central player in the complex pathophysiology of PCOS, contributing not only to metabolic aspects such as obesity but also influencing reproductive processes.

Elevated levels of leptin are consistently linked with obesity, particularly noteworthy in the context of PCOS, characterized by hyperandrogenism, inappropriate luteinizing hormone (LH) secretion, insulin resistance, and hyperinsulinemia. Leptin's involvement in PCOS contributes to the intricate interplay of hormonal and metabolic irregularities in individuals with this syndrome (Chakrabarti 2013). Hyperandrogenism, a hallmark feature of PCOS, is influenced by leptin, adding complexity to the understanding of hormonal dysregulation in PCOS.

The inappropriate secretion of LH, a key reproductive hormone, is another facet influenced by leptin in the context of PCOS. The intricate interplay between leptin and LH adds to the challenges of unraveling the exact mechanisms through which leptin contributes to the hormonal imbalance characteristic of PCOS (Rizk & Sharif 2015). Insulin resistance and hyperinsulinemia, common features in individuals with PCOS, further highlight the intricate relationship between leptin and metabolic aspects of the syndrome, emphasizing its role not only in reproductive functions but also in the broader metabolic context (Chakrabarti 2013). Leptin's involvement in PCOS extends beyond its association with obesity, encompassing intricate connections between metabolic and hormonal irregularities in the syndrome.

The relationship of PCOS with key hormonal factors such as leptin underscores the complexity of this endocrine disorder and the need for further exploration to elucidate the specific mechanisms through which leptin influences the multifaceted aspects of PCOS.

PCOS patients, characterized by hyperandrogenemia, elevated luteinizing hormone (LH) concentrations, hyperinsulinemia/insulin resistance, and obesity, provide a model to assess the inter-relationship of hyperinsulinemia and androgen excess with leptin concentrations beyond the association with obesity alone (Aversa et al., 2020).

CONCLUSION

In conclusion, the review emphasizes the intricate relationship between leptin and the pathophysiology of polycystic ovary syndrome (PCOS). The review navigates through the various risk factors contributing to PCOS susceptibility, encompassing lifestyle choices, environmental pollutants, genetic predisposition, gut dysbiosis, neuroendocrine shifts, and obesity. Despite extensive research, the exact underlying causes of PCOS remain
elusive, underscoring the need for a deeper understanding of the intricate relationship between environmental influences and genetic predisposition.

Leptin's pivotal role in energy homeostasis, regulation of reproductive hormones, and activation of ovarian enzymes positions it as a crucial player in PCOS pathophysiology. The association between elevated leptin levels and obesity in PCOS patients highlights the potential of leptin as a diagnostic marker, offering insights into the metabolic and hormonal irregularities inherent in the syndrome. All the reviewed articles contribute to the existing body of knowledge by exploring the nuanced role of leptin in PCOS development and progression. The focus on leptin's potential utility as a diagnostic marker in the clinical context opens avenues for more precise PCOS management. Future research in this field should delve deeper into the molecular mechanisms and signaling pathways involving leptin, paving the way for targeted diagnostic approaches and personalized therapeutic interventions in the realm of PCOS. The integration of novel markers, such as leptin, holds promise for advancing early detection strategies, ultimately enhancing our comprehension of PCOS pathophysiology and improving clinical outcomes for affected individuals.

Abbreviations:

1. **PCOS**: Polycystic Ovary Syndrome  
2. **ROS**: Reactive Oxygen Species  
3. **LH**: Luteinizing Hormone  
4. **FSH**: Follicle-Stimulating Hormone  
5. **LEP**: Leptin  
6. **PAI-1**: Plasminogen Activator Inhibitor-1

References


